Autoantibodies against cardiac β(1)-adrenoceptor do not affect the low-affinity state β(1)-adrenoceptor-mediated inotropy in rat cardiomyocytes

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Résumé en anglais	Circulating autoantibodies directed against the 2nd extracellular loop (EL-2) of β(1)-adrenoceptors (β(1)-AABs) have been detected in the serum of patients with various cardiovascular pathologies. β(1)-AABs induce agonistic, positive inotropic effects via β(1)-adrenoceptors (β(1)ARs). In the mammalian heart, β(1)-AR can exist in 2 distinct activated configurations (the so-called high- and low-affinity states). The aim of the present study was to investigate whether the action of β(1)-AAB is dependent on the affinity state of β(1)AR in isolated ventricular cardiomyocytes of adult Wistar rats. Immunoglobulin G (IgG) containing β(1)-AAB obtained from animals immunized with a peptide corresponding to the EL-2 of human β(1)-AR, caused a dose-dependent increase in cell shortening. Isoproterenol-induced inotropy was significantly reduced in cardiomyocytes that had been preincubated with IgG containing β(1)-AAB and in cardiomyocytes isolated from immunized rats. The negative effects of preincubation with IgG containing β(1)-AAB on the response to isoproterenol was inhibited in the presence of bisoprolol. CGP 12177A and pindolol-induced inotropy was not affected by IgG preincubation or immunization. No detectable inotropic effect of cell shortening was obtained with IgG containing β(1)-AAB in the presence of propranolol and 3-isobutyl-1-methylxanthine. The present study demonstrates that β(1)-AABs have no agonist/antagonist-like effects upon low-affinity state β(1)-ARs. This result indicates that β(1)-AABs recognize and stabilize the high-affinity state, but are unable to stabilize and (or) induce the low-affinity state receptor.
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